The Cheetah's Conservation Controversy

STEPHEN J. O'BRIEN

Laboratory of Viral Carcinogenesis
National Cancer Institute
Frederick Cancer Research and Development Program
Frederick, MD 21702, U.S.A.

The article by Merola (1994) reviews the extensive body of results that have been collected over the last decade relative to the population genetic structure of the African cheetah and the implications for survival. The author attempts to synthesize the data in a critical manner that brings into question the relevance of previous observations that the cheetah has a remarkably reduced complement of genomic variation and is suffering a physiological fitness cost as a consequence. The article revives spurious and fallacious arguments that have been made earlier and concludes that "... the genetic constitution of the cheetah does not appear to compromise the survival of the species." The end result is a rambling self-contradictory polemic that has so many misstatements, misinterpretations, disciplinary prejudices, and errors of omission as to be misleading (at best) and a disservice to the readers of *Conservation Biology*.

In hopes of addressing the major points of disagreement in a lucid manner, I shall deal only with the primary arguments and refer to previously published discussions that cover the additional points. Merola's first point argues that the levels of genetic variation found in cheetahs is not really so low because "more than 30% of compared carnivores exhibit levels of genetic diversity lower than that of cheetahs; eight of the carnivores show no polymorphism (Fig. 2)." The author continues that "rather than comparing cheetah with all other mammals, the more appropriate comparison may be other terrestrial members of Carnivora."

There are several problems with this logic. First, the criticism deals only with allozyme estimates of genetic diversity (O'Brien et al. 1983). Yet after our original observation with allozyme monomorphism, the cheetahs genomic reduction, relative to other felids and carnivore species, was affirmed with six additional measures of genomic variation (2DE gels, MHC graft exchange, MHC RFLP, mtDNA-RFLP, microsatellite and fluctuating asymmetry of cranial measurements) (O'Brien et al. 1985, 1986, 1987; Wayne et al. 1986*a*; Yuhki & O'Brien 1990; Menotti-Raymond & O'Brien 1993, 1995). Second, the estimates of lower genetic variation in eight species of carnivores (Merola's Fig. 2) derive from early allozyme surveys that are almost certainly inaccurate because they deal with very few loci, 13 in the case of *Ursus maritimus* and 21 in the case of the other 7 species (Simonsen 1982; Allendorf et al. 1979). The cheetah allozyme surveys employed 52 loci (O'Brien et al. 1983, 1987), a more robust estimate because certain allozyme loci tend to be monomorphic in all mammals (O'Brien et al. 1980; Wayne et al. 1986*b*). When "monomorphic cluster" loci are typed estimates of genetic diversity appear low because of the genes sampled. Indeed, more recent comparisons of several of the same carnivore species employing an adequate sample of 40–50 loci (O'Brien et al. 1989; Wayne & O'Brien 1987; Goldman et al. 1989) indicated appreciable allozyme variation in the same carnivora taxa that Merola's Fig. 2 report as "low." We have stated and still hold that the appropriate control would be other felid species as we have presented (O'Brien et al. 1985, 1987) and which Merola also includes in Fig. 2. Finally, later in the article Merola states that there is an "*extremely low level* of variability presently seen in cheetahs" apparently contradicting her own assertion that the cheetah has normal levels of genetic diversity. The bottom line is that six different measures of genomic diversity support

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a genetic homogenization of cheetahs' genes at the extreme low end of other Felidae and comparably studied carnivore species.

Merola's next point addresses the veracity of the cheetah's fluctuating asymmetry data. Although we agree that this measure is probably the least sensitive of the six to reflect overall genetic constitution, the results we observed using 16 cranial characters (in 33 cheetah skulls collected from three U.S. museums compared with skulls of leopard, ocelot, and maragay) showed marked fluctuating asymmetry (Wayne et al. 1986*a*). We have dealt explicitly with the statistical questions to which Merola refers elsewhere (Modi et al., 1987). We cannot be sure why asymmetry was not found by Kieser and Groeneveld (1991), but their results may be less sensitive as they examined only seven dental characters, their control species were different, and the number of specimens was a smaller data set than ours (Wayne et al., 1986*a*). In sum, we stand by our previous data, our statistical analysis, and our conclusions.

Merola then argues that the cheetah's inbreeding depression is an artifact of captivity. Although there is increasing evidence for nongenetic (e.g., management, nutritional, behavioral) components that affect cheetah survivorship and reproduction, to exclude the influence of the genetic components is to ignore overwhelming evidence. For example, the sperm abnormalities that are now known to affect fertility (Howard et al. 1993) are identical in free-ranging cheetahs and in captive cheetahs (Wildt et al. 1983, 1987). Second, the high mortality estimates (>90% reported by Caro and Laurenson [1994]) due to lion and hyena predation in the Serengeti are likely inflated for two reasons. First, cub deaths due to predation are not independent estimates because once a cheetah den is discovered, all the cubs will be taken regardless of litter size. Second, it is difficult to exclude observer influence since a researcher in a vehicle watching a cheetah den would alert lions and hyenas to their subject. These caveats raise serious questions about the interpretations of the Serengeti study that dismiss the role of the cheetah's genetic status in their survival.

Even if one concludes that ecological factors are primary regulators of cheetahs in the Serengeti, this does not mean that the physiological factors impaired by a history of inbreeding would not pose a problem in a different time or ecosystem. Finally, the immunological/virological criticism reflects a misunderstanding of the concept of coevolution of host and parasite genomes (O'Brien & Evermann 1988; Heeney et al. 1990). Merola's argument about the proximity of cheetahs in the feline infectious peritonitis virus (FIPV) epizootic is so speculative that it makes little sense other than to provide dangerously false hope based on woeful ignorance of the kinetics of coronavirus spread in free-ranging populations.

The cheetah story has been carefully addressed in previous articles, although only recently have cumulative data been reviewed and the criticisms addressed (O'Brien 1994, in press; O'Brien & Evermann, 1988). The evidence for genetic reduction in cheetahs is extensive, covering several categories of loci and the most appropriate control species. All cat species including the cheetah's nearest relative, the puma (*Felix concolor*), have abundant genetic variation in contrast to cheetahs (Roelke et al. 1993; O'Brien et al. 1985). Cheetahs are hard to breed, have reproductive and congenital abnormalities, and have an extremely high cub mortality both in captivity and in a natural setting. The potential for a homogeneous response to a deadly pathogen, FIPV, was realized in a epizootic that was unprecedented in extent in other felid species (Heeney et al. 1990). The data on the cheetah's genetic structure and physiological status are extensive and persuasive. The decay of habitat is certainly a primary threat to cheetahs today, but to ignore the lesson on the cheetah's genetic history and conclude that genetic consideration are "misdirected" is both ideological and inaccurate.

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